ACUTE RENAL FAILURE FOLLOWING ABDOMINAL AORTIC ANEURYSM RESECTION

John Alan Fox

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ACUTE RENAL FAILURE FOLLOWING
ABDOMINAL AORTIC ANEURYSM RESECTION

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John Alan Fox

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Dedicated to my parents--John and Esther Fox--
who have shared my peaks and valleys and who have always been a source of understanding and encouragement: May they be as proud of me as I am of them.

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Introduction

In surgery, more so than any other medical subspecialty, doctors are required to make their patients sicker before they can make their patients better. Besides treating the gamut of purely medical diseases that a patient will have either prior to or as a result of his surgical disease, the surgeon must also deal with the effects that anesthesia and operation have on the patient's condition. It is this aspect that makes surgery perhaps the most challenging of the clinical sciences.

No group of patients is more challenging to the surgeon than the group that presents with or acquires as a post-operative complication, acute renal failure(ARF). As a whole, ARF in surgical patients has mortalities reported as 50 to 90% depending on the surgical disease. Contributing to this high mortality is the fact that renal shutdown is often only part of the multiple organ failure that makes these patients particularly difficult management problems.

Because ARF carries a high mortality in surgical patients, the purpose of this study was to identify risk factors that lead to ARF, predict a subset of surgical patients that are at great danger of developing renal failure, and thereby suggest steps that could be taken to prevent the syndrome. This project was designed as a retrospective study of ARF in abdominal aortic aneurysm patients. By selecting a specific surgical disease, in this case a disease in which development of ARF is a significantly poor prognostic sign, an attempt was made to choose a patient population in which the pre-operative status, operative procedure, and post-operative management were generally comparable. In

this manner, the variability between "non-renal failure" and "renal failure" groups would hopefully be minimized.

The following general questions were asked:

(1) Does there exist in patients post-aneurysmectomy a syndrome of renal dysfunction that is different from the picture of full blown renal failure? This renal dysfunction might be marked by a transient rise of serum creatinine and BUN that is not necessarily outside the normal range. By studying a group of patients such as this, in which changes in renal function were subtle rather than catastrophic (and subsequent clinical picture simple rather than complex), it was hoped that risk factors pre-disposing to changes in renal function would be easier to identify.

(2) Is hypotension a singular factor which pre-disposes these patients to ARF or is there a combination of hypotension plus a pre-operative factor that appears commonly in ARF patients?

(3) Is there any correlation between intra-operative parameters and renal failure?

(4) Finally, is there any post-operative non-renal complication that specifically correlates with the development of renal failure?

Literature Review

Mortality rates for renal failure patients are reported in the literature in three types of studies that differ depending on the patient population. These studies involve surveys of: a) surgical, medical, and obstetrical patients, b) patients who are immediately post cardiac surgery, and c) abdominal aortic aneurysm patients (who often develop a "sequential systems failure" after aortic repair of which acute renal failure is one aspect of their illness).

Analyses of the first type all emphasize one specific point: acute renal failure in patients immediately after a major surgical procedure carries a higher mortality than the same syndrome in medical or obstetrical patients. Kennedy et al,[17] who studied renal failure in 62 obstetrical, 56 medical, and 133 surgical patients over an 11 year period, reported mortality rates of 12%, 35.7%, and 58% respectively, for the three groups. Overall mortality was 44%. Additionally, these investigators reported a decrease in the number of patients admitted to their renal unit in the latter years of the study but they reported an increase in the percentage of surgical patients. Stott et al,[22] in a similar study of renal shutdown, gathered 37 "general surgical patients", 18 cardiovascular surgical patients, 14 "undiagnosed obstructive uropathy" patients, and 30 medical patients and obtained mortality rates of 70%, 61%, 57%, and 47% respectively, over a 27 month period.

Studies on open heart patients continue to confirm the increased mortality risk that renal failure brings. One study of 509 consecutive survivors of open heart procedures obtained 198 (39%) patients who had

"renal dysfunction" post-operatively.[5] (Renal dysfunction here was described as a post-operative serum BUN>50mg/100ml or a post-operative serum creatinine> 2.0mg/100ml.) The average high BUN and creatinine for these patients post-operatively was 90.2±52.0 and 3.8±1.4, respectively. Mortality in the "renal dysfunction" group was reported at 30.3% as compared to 1.6% of the normal renal group. Bhat et al[7] reported a 16% mortality rate in cardiac surgery patients who acquired renal failure and a 0.9% death rate in those who did not. They divided their renal dysfunction group into patients whose peak post-operative serum creatinine levels were: a) between 1.6 and 1.9mg/100ml, b) between 2 and 5mg/100ml, and c) greater than 5mg/100ml and coincident mortality was 7% for subset "a", 8% for subset "b" and 66% for subset "c".

Reports of renal insufficiency following abdominal aortic aneurysm resection have been confined largely to patients who have undergone emergency resection of ruptured or expanding aneurysms. These studies are so numerous that one gets the impression that acute renal failure must be uncommon in elective resections. Tilney _et al_,[23] developed the concept of "sequential organ systems failure" in a review of 18 ruptured abdominal aortic aneurysm patients who, after renal shutdown, went on to develop, in sequence, pancreatic, pulmonary, hepatic, central neurologic, gastro-intestinal, and cardiac failure. Only 1 of these 18 patients survived despite maximal antibiotic, ventilatory, and renal support. Renal support consisted of hemodialysis for 4-6 hours every second day.

In another study of the Yale-New Haven Hospital experience of ruptured aneurysms from 1956 through 1969,[25] 69% of the patients who survived operation but succumbed later in the post-operative course had documented ARF. Patients who ultimately left the hospital had only a 22% incidence

of documented ARF. Furthermore, in these patients with ruptured aneurysms, 79% of those who presented with renal compromise before operation (as defined by BUN>20mg%) died. No mention is made of the number of these patients who acquired ARF post-operatively.

Other series confirm the unusually high mortality of the ARF/ruptured aneurysm group. Graham et al[14] attribute a renal factor as a cause of death post-operatively in only 3 of 36 patients; however, in their collection of 107 ruptured aneurysm patients, 22 died intra-operatively (14 of these had known oliguria before the procedure). Only a single oliguric patient survived surgery. In another study,[11] 4 of 5 patients who died from one to five days post-operatively were anuric as were 9 of 11 patients who died six to sixty days after surgery. No patients (8 total) who required peritoneal dialysis in this study survived. Overall mortality rates in these studies were 54%[14] and 60%.[11]

Although ARF is found most often in patients with ruptured aneurysms, the outlook when it occurs in elective resection is just as grim. Couch et al found nearly a 6 % incidence of anuria following elective resection (4/62) with an associated 50% mortality.[10] Another study, which included 6 patients who developed ARF following elective resection,[8] showed an 83% (5/6) mortality in these patients. Anuria in ruptured aneurysms in these studies carried mortalities of 100% (9/9)[10] and 88% (7/8)[8].

This high mortality associated with post-operative ARF suggests the need for effective prophylaxis. (The surgeon can take a more active role in instituting preventative measures intra-operatively as compared to pre-operatively where often there is little time to maximize the patient's status.) Intra-operative preventative measures that have been advocated to decrease the stress on the kidneys during aneurysm resection include

infusion of mannitol and aggressive maintenance of intra-vascular volume. Post-operative administration of adequate nutritional fluids has been advocated.

Mannitol is often infused into the patient either immediately after the operation has begun or after the aorta has been cross-clamped in an attempt to maintain renal perfusion. This osmotic diuretic has been shown to increase glomerular filtration rate and urine flow before and after cardiopulmonary bypass in open heart patients[12]. Use in abdominal aortic aneurysm procedures has been rationalized on the basis of an experimental model in dogs. In this model, aortic cross-clamping below the renal arteries resulted in ischemia of the renal cortex as determined by radioactive xenon washout techniques[3]. If mannitol was infused directly into the renal artery after aortic occlusion, the renal cortical ischemia was eliminated[2].

Intravascular fluid therapy to maintain urine flow intra-operatively and prevent profound hypotension following aortic de-clamping were two factors stressed by Thompson <u>et al</u>[24] in a study of 670 elective resections. In these patients, infusion of a "balanced salt solution" (5% Dextrose in Ringer's Lactate) during the procedure maintained urine output at a rate of 2.1 cc/min. and produced a drop in systolic blood pressure after declamping at an average of only 18 mm Hg. Only 2 patients in this study (0.3%) developed renal insufficiency post-operatively. (Both died.)

Once the diagnosis of ARF is established, the treatment is usually focussed on two major areas: hemodialysis and nutritional support.

Kleinknecht's group[18, 19] extensively studied 760 medical, surgical, and obstetric patients in ARF and divided their patients into two groups depending whether or not aggressive hemodialysis was employed. Surgical

patients had a 54% mortality without dialysis and a 42% mortality with dialysis, and the overall incidence of death due to gastrointestinal hemorrhage was reported to decrease from 32% to 8% with dialysis.

Abel et al[4], in a randomized prospective study of medical and surgical patients who developed ARF following renal ischemia or exposure to nephrotoxic substances, produced a 75% survival rate in patients who received (via superior vena cava) a hyperalimentation solution consisting of essential L-amino acids and hypertonic glucose. Patients who received an intravenous solution without the amino acids had a 44% survival. Additionally, these authors maintained that the serum BUN value for "treated" patients stabilized sooner than the "non-treated" patients implying that hyperalimentation with L-amino acids and glucose not only slowed the rise in BUN by decreasing catabolism but may also have shortened the duration of the ARF. Survival was even more impressive when patients who were dialysed were considered as 11 of 17 patients survived who received the "renal failure fluid" in addition to hemodialysis while only 2 of 11 survived who received the control solution and were hemodialyzed.

Data regarding ruptured aortic aneurysm patients is more muddled. One study, of only 9 patients who developed ARF following aneurysm rupture, claimed a survival rate of 66% with hemodialysis therapy without special hyperalimentation treatment[9]. Abel et al[1], in a sequel to their original report mentioned above, studied ARF only in patients with ruptured aortic aneurysms and produced only a 12.5% survival rate (4 of 32 patients) despite essential l-amino acid hyperalimentation and aggressive hemodialysis.

Except for Abel et al's initial report[4], ARF in the surgical patient represents at least a 40% mortality rate despite treatment. This high mortality associated with post-operative ARF underscores the importance

of predicting patients who are at risk for ARF so that preventative measures may be undertaken.

Methods

Patient Population

The records of 131 patients who had undergone resection of either a ruptured or non-ruptured abdominal aortic aneurysm at Yale-New Haven Hospital or at the West Haven Veterans Administration Hospital in the time period 1974-1978 were reviewed in detail. Of these 131 patients, 10 succumbed prior to having a serum creatinine drawn post-operatively and were considered separately. The remaining 121 patients were divided into four groups as follows:

Group I was defined as the group having "normal" renal function post-op. These patients had a post-op serum creatinine of less than 1.5mg%. Their highest creatinine post-op was less than a 0.5mg% increase over pre-op values.

Group II included patients whose highest serum creatinine was greater than 1.5mg% but whose change in creatinine was (like Group I) less than 0.5mg% over pre-op values. This group was felt to represent patients with some abnormality in renal function but who were unaffected by operation.

Group III was defined as showing some form of "renal dysfunction" as a result of operation and included patients with normal serum creatinine levels throughout the post-op period (creatinine less than 2.0mg%) but whose highest post-op creatinine was greater than or equal to a 0.5mg% increase over pre-op levels.

Group IV included patients with "renal failure" as a result of surgery and encompassed all patients whose post-op creatinine

reached levels greater than or equal to 2.0mg% and who showed at one point post-op an increase in creatinine greater than or equal to 0.5mg% over pre-op values.

The above criteria defining each group were arbitrarily set in order to select patients who had a) Normal renal function pre- and post-operatively (Group I), b) No change in renal function post-operatively but with a borderline level of renal function pre-operatively (Group II), c) A subtle change in renal function post-operatively but with normal renal function tests through out (Group III), and d) ARF post-operatively (Group IV). The key value in grouping these patients was serum creatinine. By definition, all patients in this study with a rise in serum creatinine 0.5mg% post-operatively fell into Groups III or IV. A rise $<$o.5mg% post-operatively placed the patients in Groups I or II. In some cases, particularly emergency resections, a pre-operative creatinine was not obtained. To classify these patients, the highest and lowest post-operative creatinines were considered and if the difference between them was $\geq$0.5mg%, the patient was classified in Group III or Group IV. Pre-operative creatinines were unavailable in only 4 patients in Group III and 6 patients in Group IV and this latter method was used to classify these 10 patients.

The patients were defined as being "hypotensive" if a recorded systolic blood pressure of less than or equal to 90 mm Hg was noted anytime during the pre-operative evaluation, operative procedure, or during the post-operative course.

Pre-Operative Evaluation

All patients were evaluated to determine the existence of any factors in their baseline state that were felt to increase the risk for development of acute renal failure.

The aneurysmectomy was classified as either "emergent" if the patient was rushed to the operating room with the diagnosis of ruptured or expanding aneurysm or "elective" if the patient was scheduled for operation on a certain day and came to operation on that day with an uneventful pre-op course.

The presence or absence of diabetes mellitus, liver disease (by history of chronic alcohol abuse or biopsy proven cirrhosis), hypertension (by history), myocardial infarction (also by history), and age were all noted from the admission work-up. Also noted from the admission work-up were any factors that might contribute to a suboptimal baseline renal status. Classified (in this study) as pre-existing "renal disease" were a history of documented glomerular or interstitial kidney disease, history of renal stones or gout, and history or urological procedures (nephrectomy, ureteral surgery) excluding prostatectomies.

Additionally, a rough assessment of nutritional status was made by recording the heights and weights of each patient from the anesthesia records. These values were then compared to the Metropolitan Life Insurance Tables for average Weights for Men and Women According to Height and Age and the number of kilograms either above (+) or below (-) these averages were recorded for each patient. For patients greater than 69 years old, the values for ages 60-69 on the Metropolitan Life Tables were use.

Operative Parameters

The operative repair in all cases was essentially the same. The procedure consisted of first exteriorizing the small bowel and then opening the posterior parietal peritoneum over the aneurysm. Once proximal and distal control of the aneurysm was achieved, the patient was then systemically heparinized and the aneurysm was cross-clamped proximally at its

neck (which was usually beneath the renal arteries) and distally at its extension to the iliac arteries or, in some cases, the femorals. The aneurysm was then opened and the intraluminal clot removed; back-bleeding lumbar arteries were oversewn and Dacron bifurcation grafts sewn in first proximally and then one distal limb. The proximal aortic cross clamp and one distal clamp were then removed, re-establishing flow to one leg. Following completion of the second distal anastomosis and clamp removal, heparinization was reversed with protamine, hemostasis achieved, and the abdomen closed.

Variability in the intra-operative management occurred in two areas. Some surgeons infused the patients continually with a 20% solution of mannitol to maintain urine output; others did not. Secondly, certain operators injected several milliliters of a 0.1% solution of Neosynephrine in the distal limbs of the graft at the time of aortic declamping. This was done to minimize the hypotension associated with re-establishment of limb flow. The use of these drugs was determined from the anesthesia and operative records.

These records were also used to determine anesthesia time, urine output, units of blood products (whole blood, fresh frozen plasma, and packed red blood cells) infused, and renal artery compromise (either by aneurysmal involvement or cross-clamping briefly above their take-off) during the operation.

Aortic cross clamp time was difficult to record as often the time of proximal clamp placement was noted but not that of proximal clamp removal. Protamine administration was recorded at a time corresponding to removal of the second distal clamp. In order to record one constant time, aortic cross clamp time was defined here as that time between proximal

aortic clamping and protamine administration (complete de-clamp of the aorta). This definition of aortic cross clamp time includes time that was used for sewing the aortic bifurcation graft in place along with time used for achieving hemostasis at the end of the operation.

Post-Operative Course

The post-operative period was studied in all patients but more intensively so in those patients with either renal failure or renal dysfunction in hopes of: a) evaluating the incidence of a multiple organ systems failure syndrome and b) correlating an associated complication as either a sequelae or as a cause of the renal problems. The events surrounding a patient's demise deserved and got greater scrutiny. While systematically reviewing this data, it was noted that several patients developed jaundice post-operatively and this data is presented in Chart VI. Patients were defined as being hyperbilirubinemic if either their highest recorded post-operative direct bilirubin was greater than or equal to 0.5mg% or if their highest post-operative total bilirubin was greater than or equal to 1.5mg%.

Results

Chart I represents data on which the patients in this study were divided into Groups I-IV. Of the 121 cases for which renal status could be evaluated following surgery, 97 cases had no post-operative change in renal function while 24 cases showed some evidence of renal dysfunction. Ninety patients met criteria of Group I, having essentially normal serum creatinine levels throughout their hospitalization. There were 7 patients in Group II with an average pre-op creatinine slightly elevated (2.2 ± 0.48mgms%) but with an average high post-op creatinine (2.2 ± 0.48mgms%) unchanged ($p > 0.5$, Student t test) from the pre-op value. These patients had a low post-op creatinine averaging 1.3 ± 0.75mgms%. Eleven patients were classified in Group III and were defined by this study as having "renal dysfunction" as a result of surgery. Pre-operative creatinine in this group averaged 1.01 ± 0.19mgms% with an average high post-op creatinine of 1.60 ± 0.20mgms% ($p < 0.001$). Finally, 13 patients had "renal failure" (Group IV) with creatinines averaging 1.7 ± 0.40mgms% before surgery and having a high post-operative creatinine averaging 7.2 ± 5.4mgms% ($p < 0.001$).

Pre-Operative Risk Factors

Chart II summarizes the pre-operative status for each of the four patient groups. Pre-existing liver disease or diabetes mellitus was uncommon in the aneurysm patients studied. Diabetes was seen in only 3 patients--2 patients with normal renal function tests (Group I) and 1 patient who developed renal dysfunction (Group III). Liver disease

was seen in only 5 patients--4 patients in Group I and 1 in Group III.

Renal disease occurred in 21 patients--13(14%) in Group I, 5(71%) in Group II, and 3(23%) in Group IV. No patients in Group III had any history of pre-existing renal disease. These incidences were significantly different ($p < 0.01$) between Group I and Group II ($X^2=10.44$) and between Group II and Group III ($X^2=7.61$) but were not significant to $p < 0.05$ between any of the other groups. The incidence of renal disease in patients who had no change in renal function after surgery (Groups I + II) was 18,5%(18/97) while the same incidence in Groups III + IV (patients with either renal dysfunction or renal failure post-operatively) was 12.5%(3/24). These incidences were not statistically different to $p < 0.05$ according to the Chi Squared Test.

Of the total 21 patients with renal disease, 9 had a history of renal stones, 4 had diagnosed gout, and 4 had previous urological surgery--3 with nephrectomies and 1 with a ureteral resection for an unrecorded reason. Of the remaining 4 patients, 1 had congenital absence of her left kidney; one had recent onset of 4+ proteinuria and was found to have a left renal artery occluded by his abdominal aortic aneurysm; one was admitted for work-up of hematuria (which was negative) and was found to have an abdominal aneurysm at this admission; and, finally, one patient had a kidney disease of unexplained etiology with an admission creatinine of 3.2 mgms%. Of 3 patients with "renal disease" who developed ARF (Group IV), one had renal stones, one had congenital absence of the left kidney, and one had a right nephrectomy over 40 years prior to admission for a "hypernephroma."

The incidence of hypertension was highest in Group II(71%) followed by Groups III(64%), IV(46%), and I(29%) and was statistically significant ($p < 0.05$) only between Groups I and II ($X^2=3.92$). Hypertension

in the combined groups I + II vs III + IV calculated as 32%(31/97) and 54%(13/24), respectively. These incidences were not significant at p=0.05 (X^2=3.19).

Myocardial infarction occurred with incidences of 36%(32/90), 29%(2/7), 9%(1/11), and 38%(5/13) in Groups I, II, III, and IV, respectively. Inter-group comparisons of these frequencies were not statistically significant (Chi Squared, p=0.05) nor was the comparison between I + II(34/97=35%) vs III + IV(6/24=25%).

Ruptured aneurysms occurred with a frequency of 11%(I), 0%(II), 45%(III), and 61%(IV). The incidence of rupture in Group IV was statistically higher (p=0.05) from the incidence of rupture in all the other groups (X^2=16.69 vs Group I, 4.84 vs Group II, and 22.83 vs Group III). Additionally, the frequencies were significantly different to p=0.05 when comparing Group I vs Group III (X^2=6.63) but were not significantly different when comparing any other groups.

Age was not statistically different between any of the four groups ($p < 0.1$, Student t test) with the average age (in years) being 68 ± 8, 72 ± 8, 66 ± 6, and 70 ± 8 for Groups I, II, III, and IV, respectively.

Intra-Operative Parameters

Data collected regarding variables occurring during surgery appears in Chart III. Patients who later developed renal failure (Group IV) had significantly longer operations (354 ± 128 vs 282 ± 85 minutes, $p < 0.01$), infusion of more units of blood products (13.3 ± 10.1 vs 6.0 ± 4.0 units, $p < 0.001$), a higher incidence of documented renal artery compromise during surgery (31% vs 3.3%, $p < 0.01$) and a lower average urine flow rate during the operation (1.27 ± 0.84 vs 2.64 ± 1.68 cc/minute, $p < 0.05$) than the patients who had no abnormalities in renal function (Group I).

Aortic clamp times were not significantly different (96 ± 47 vs 79 ± 34 minutes, $p < 0.2$) between Group IV and Group I. Anesthesia time, aortic clamp time, blood product infusion, urine flow rate, and renal artery compromise were not statistically different between Group IV and Group II (patients with abnormal pre-operative serum creatinines) or Group III (patients with "renal dysfunction") with the exception of intra-operative blood product infusion between Group IV and Group II. The renal failure patients (Group IV) received more blood during surgery than the patients of Group II (13.3 ± 10.1 vs 5.4 ± 3 units) to $p < 0.05$.

Mannitol was used in 69% (62) of patients in Group I, 57% (4) of patients in Group II, 45% (5) of patients in Group III, and in 8 (61%) of patients in Group IV (None statistically different to $p < 0.05$, Chi-Squared) while neosynephrine was injected into the distal limb of the grafts before aortic de-clamping in 22% (20), 43% (3), 27% (3), and 61% (8) of the patients in Groups I, II, III, and IV, respectively (None statistically different to $p < 0.05$, Chi Squared except Groups I vs IV where $X^2=6.99$).

Hypotensive Patients

Of the 97 patients in Groups I and II, 32 (or 33%) had a pre-intra-, or post-operative hypotensive episode while, of the 24 patients in Groups III + IV, 17 (or 70%) were hypotensive sometime during their hospitalization ($X^2=10.80$). Chart IV summarizes pre-operative data in these patients. There was no difference between the weight/height ratio ($p > 0.5$, Student t test) or in the incidence of renal disease (15.6% vs 12%--$p < 0.5$ Chi Squared). Hypotensive patients who had no change in renal status post-operatively averaged 2.98 ± 9.42 kilograms below expected weight for height and age while patients who developed

renal changes averaged 3.4±8.46kgms below expected weight for height and age.

There was a statistically significant ($p < 0.05$, Chi Squared) higher incidence of hypertension (53% vs 18.8%, $X^2=4.61$), emergency resection (65¢ vs 12.5%, $X^2=11.89$), and mortality (28% vs 3%, $X^2=7.80$) in the renal compromised group but a higher, statistically insignificant ($p > 0.05$, Chi Squared) rate of myocardial infarction in the combined Groups I + II (37.5% to 12%, $X^2=2.45$).

Chart V compares operative parameters among the hypotensive patients. The renal malfunction Group III + IV had significantly more transfusions of blood products during surgery (12.2±8.1 vs 5.9±3.7 units, $p < 0.001$, student t test), a statistically higher incidence of renal artery compromise during surgery (24% vs 0%, $p < 0.025$, Chi Squared) and a statistically lower urine flow rate during surgery (1.3±0.83 vs 2.4±1.4 cc/minute, $p < 0.01$, student t test) than the patients in Group I + II who did not develop a change in renal function following surgery. Anesthesia time (340±135 vs 276±99 minutes), aortic clamp time (98±52 vs 81±30 minutes), and mannitol treatment (59% vs 53%) was not statistically different between the Groups III + IV and I + II.

Hyperbilirubinemic Patients

While considering each patient's post-operative course, it became evident that a fairly high number of patients developed an abnormality in their serum bilirubin levels. Several patients even developed severe jaundice. Chart VI compares the hyperbilirubinemic patients of Groups I + II (normal renal function) with the patients of Groups III + IV (renal malfunction group). From these data, it is clear that patients with renal compromise are more likely ($p < 0.25$, Chi Squared) to have

associated liver dysfunction (54% incidence vs 15.5% incidence) however the severity of the insult to the liver was not significantly different ($p > 0.5$ for both direct and indirect bilirubin values, Student t test) when peak bilirubin levels are considered. The ratio of direct bilirubin to total bilirubin was $1.87 \pm 1.31/3.89 \pm 2.1$ mgm% for Groups I + II and $1.96 \pm 2.0/4.23 \pm 3.7$ mgm% for Groups III + IV. There was also no difference in the duration of operation (328 ± 127 vs 344 ± 146 minutes, $p \geq 0.5$, Student t test).

What did differ between the hyperbilirubinemic patients with and without renal changes following surgery was total amount of blood products given intra- and post-operatively ($p < 0.025$, Student t test), hypotensive indices ($p < 0.5$, Chi Squared), incidence of emergency resection ($p < 0.1$ Chi Squared), and mortality rate ($p < 0.25$, Chi Squared). The renal compromise group received an average of over 26 units of blood products during their hospital stay (to over 14 units in the other group). 77% of the renal group had a hypotensive episode (compared to 47% of the normal group) and the incidence of emergency resection was 61% for the renal dysfunction patients (vs 27% for the normal patients). None of the patients in Groups I + II died while 3 of the renal group who were hypotensive died.

Mortality

Deaths occurred only in Groups I (1%--1/90) and IV (46%--6/13). The single death in Group I was attributed to a sudden, massive anterior wall myocardial infarction on Day #4 post-operatively in a course that was otherwise normal following surgery. Chart VII presents a profile of all 6 acute renal failure patients that did not survive and illustrates a wide variance in pre-operative and intra-operative factors found in this

small group of patients. Of all the factors listed in Chart VII, it should be noted that 4 of 6 patients (67%) presented with signs of a ruptured aneurysm. Additionally, all except Patient #4 had some type of infection of either the bloodstream or the lungs post-operatively. Three patients were either peritoneally or hemodialyzed and they represented the only patients who were so treated in this study. Hyperalimentation as treatment for acute renal failure was not instituted.

Peri-Operative Mortality

Ten patients were not included in the previous data because they did not survive surgery long enough to have their post-operative renal status checked (i.e., no post-operative serum creatinine was recorded). Of these 10 patients, 8 presented with profound hypotension and acute abdominal pain and were found to have a ruptured aneurysm at operation. Seven of these eight died intra-operatively of either hemorrhage or cardiac damage secondary to the shock. One patient survived surgery but remained anuric and died of a cardiac arrest soon after surgery. A single patient of these 8 had "renal disease" in the form of gout. Of the two elective resection patients, one (with labile hypertension) suffered 2 hypotensive episodes intra-operatively and arrested immediately after arriving in the surgical intensive care unit and the second (with a history of nephrotic syndrome but on prednisone and with normal renal function tests pre-operatively) developed refractory ventricular fibrillation at the end of the procedure and died intra-operatively. Average age of all 10 patients was 73.2 ± 8.6 years.

Total mortality for the 131 patients reviewed in this series was 13% (17/131). Elective resections carried a 5% mortality (5/100) with

2 of these 5 mortalities in patients with post-operative acute renal failure. Ruptured aneurysms carried a 39% mortality (12/31) and of these 12 deaths, 4 occurred in patients with an acute renal failure syndrome.

Discussion

This study was undertaken when it was noted from personal experience on the wards of Yale-New Haven Hospital that surgical patients with acute renal failure present major problems in management and, as noted repeatedly in the l terature, have an extremely high mortality. By studying a group of patients at risk for ARF, it was hoped that predictive factors that correlate with either onset or final outcome of this complication could be defined. In this study, no single factor could be identified as a necessary and sufficient cause of acute renal failure following abdominal aortic aneurysm resection.

As Chart II illustrates, acute renal failure patients (Group IV) showed no statistical difference from patients who had normal renal function tests (Group I) in the pre-operative incidence of renal disease (X^2=0.16, $p < 0.75$), diabetes mellitus (X^2=0.28, $p < 0.75$), liver disease (X^2=0.00006, $p < 0.9$), myocardial infarction (X^2=0.01, $p < 0.95$), hypertension (X^2=0.88, $p < 0.5$), or age (t=1.07, $p < 0.4$). Acute renal failure patients did have a statistically significant higher incidence of emergency resection (X^2=16.69, $p < 0.005$) and death (X^2=29.92, $p < 0.005$) than the patients who had normal renal function. In evaluating this pre-operative data, two areas deserve further discussion.

First, patients with pre-existing renal disease prior to aneurysmectomy would logically be thought to be at higher risk for developing ARF post-resec.ion. The data presented above, which showed no difference in the incidence of renal disease between ARF patients (Group IV) and "normal" patients (Group I), coupled with the 7 patients in Group II

(who had abnormal serum creatinine values and a high incidence of "renal disease" before surgery but who did not develop ARF after surgery), would imply that underlying renal malfunction may not necessarily represent a major risk factor in acquiring an ARF clinical picture. It should be noted, however, that "renal disease" in this study was an all-inclusive definition of kidney (interstitial and glomerular) disease and urological (ureteral and nephrolithical) disease and that the sub-group of patients with "kidney disease" may indeed be at higher risk for ARF post-aneurysmectomy. Additionally, the pre-operative serum creatinine values in Group IV were statistically higher than the pre-operative serum creatinines in Group I ($t=4.34$, $p<0.001$) implying that several patients in Group IV who are thought to have "normal" renal function may be unaware of an existing kidney disease. The possibility that the observed incidence of "renal disease" in Group IV may be higher than recorded cannot be excluded.

Secondly, because there is a statistically higher incidence of emergency resection in the patients of Group IV, it was thought that hypotension associated with this type of resection may correlate well with development of ARF. The data in Chart IV studies this hypothesis.

Hypotension occurred in a significantly higher incidence ($X^2=10.80$, $p<0.001$) of patients who had some change in renal function post-operatively (Groups III + IV--17/24 patients, 71%) than in those patients who had no change in renal function after surgery (Groups I + II--33/97 patients, 34%). Pre-operatively, there was no difference in the incidence of renal disease ($X^2=0.003$, $p<0.9$), myocardial infarction ($X^2=2.45$, $p<0.25$), or Weight/Height Variance ($t=0.13$, $p<0.5$) between hypotensive patients with no change in renal function post-op (Group I + II) and hypotensive patients with a change in renal function post-op (Group III + IV). The hypotensive renal malfunction group did have a higher incidence of

emergency resection ($X^2=11.89$, $p < 0.005$) and hypertension ($X^2=4.61$, $p < 0.05$) than the group that had no change in renal function tests after surgery.

Intra-operatively, (Chart V) hypotensive patients who developed renal dysfunction had a statistically higher number of blood product transfusions ($t=3.72$, $p < 0.001$), an increased incidence of renal artery compromise ($X^2=5.36$, $p < 0.025$), and a decreased urine flow rate ($t=2.96$, $p < 0.005$) than hypotensive patients with no change in renal function after surgery. Mannitol use ($X^2=0.17$, $p < 0.9$) and aortic clamp time ($t=1.29$, $p < 0.4$) were not statistically different in these groups.

The existence of 33 patients who were hypotensive during some time in their hospital course implies that this factor by itself is not a necessary and sufficient cause of ARF. The duration of hypotension was difficult to quantify from the patient records but the patients classified as hypotensive were in this state long enough to either warrant treatment with vaso-pressive agents or to produce a renal ischemic episode. This study defined hypotension as a blood pressure <90 mm Hg diastolic and it is possible that the renal malfunction patients had a hypotensive episode that was more severe than the episode of the non-renal malfunction patients.

Intra-operatively (Chart III), the patients in Group IV had longer operations (anesthesia time: $t=2.65$, $p < 0.10$), infusion of more units of blood products ($t=4.72$, $p < 0.001$), a higher incidence of renal artery compromise ($X^2=9.51$, $p < 0.001$), and a lower urine flow rate ($t=2.16$, $p < 0.05$) during surgery than the patients in Group I. Aortic clamp times were not significantly different between Group I and Group IV ($t=0.30$, $p < 0.5$).

Couch et al[10] presented data from which they maintained that mortality in aneurysm patients undergoing both elective and emergency resections was not affected by the intra-operative parameters of anesthesia time, aortic clamp time, or blood product infusion. However,

their study did show that 7 of 8 patients with ruptured aneurysms who received $<$20 units of blood ultimately died. The number of these patients who also had acute renal failure was not given, making a comparison between their series and this series difficult. It should be noted that 9 patients with ruptured aneurysms in the series of Couch et al had acute renal failure and died so it is reasonable to assume that a number of the 7 patients who received $<$20 units of blood and died also died with ARF.

Of the intra-operative parameters studied in this series, anesthesia time, administration of blood products, and renal artery compromise are parameters that are not easily varied by the surgeon. Urine flow rate, on the other hand, can usually be maintained by repletion of intravascular volume and by administration of diuretics intra-operatively. Thompson et al[24] were able to achieve a urine flow rate during surgery of 2.1 cc/min. by closely monitoring the intra-vascular volume and replacing losses with a 5% Dextrose/Ringer's Lactate solution and this value compares well with the urine flow rate of Group I. The mortality in Thompson et al's study was 0.3%.

The osmotic diuretic mannitol, of which administration during abdominal aortic aneurysm resection can be based on an experimental animal model[2,3], was given in a statistically comparable number of cases in Groups I and IV (X^2=0.05, p $<$0.9) and afforded no protection against ARF for the 8 patients of Group IV in which it was used. Although a closer study of these 8 patients is not presented here, the development of renal failure in them would imply that mannitol has certain limitations in its protective properties on the kidney. These limitations may involve the time (either pre-, post-, or during aortic clamping) at which the diuretic was given, the degree of renal damage that had already occurred before administration, or an entire host of factors (be they nephrotoxic

drugs used concurrently with the mannitol or the pre-existing baseline status of the patient) that are outside the realm of this study.

In an attempt to maximize the chances of selecting factors predictive for ARF, patients undergoing aneurysm resection who had subtle changes in their creatinine levels post-operatively were searched for. These patients, who would have a form of "renal dysfunction" not as serious as the clinical picture of ARF, were assumed to be a "pre-renal failure" (or renal insult) group. The concept of post-operative "renal dysfunction" comes from a study by Bhat et al[7] of open heart patients, several of whom had rises in serum BUN and creatinine to borderline normal levels without signs of oliguria. This change in renal function was speculated to be due to absence of pulsatile flow during cardiopulmonary bypass or to subtle changes in pressure during the pump run and, generally, the "renal dysfunction" resolved after several days.

Eleven patients in the current study met criteria for renal dysfunction (a rise in serum creatinine $>$ 0.5 mgms% from pre-operative values). This represented only 9% of the patients studied. The majority of patients either maintained normal renal function tests following surgery or developed obvious renal failure. Selection of these 11 patients may have been biased as 4 patients were placed in Group III by subtracting low post-op serum creatinine levels from high post-op serum creatinine levels because no pre-op serum creatinine value was recorded.

The renal dysfunction patients in this study (Group III) had no significant difference in incidence of myocardial infarction (X^2=2.03, $p < 0.25$), renal disease (X^2=0.11, $p < 0.75$), or age (t=0.52, $p < 0.5$) than the patients with normal renal function (Group I). The incidence of hypertension (X^2=3.92, $p < 0.05$ and emergency resection (X^2=6.63, $p < 0.01$ was higher

in Group III than in Group I. Anesthesia time ($t=0.17$, $p<0.5$), aortic clamp time ($t=0.56$, $p<0.5$), mannitol use ($X^2=1.48$, $p<0.25$), blood products infusions ($t=0.47$, $p<0.5$), renal artery compromise ($X^2=1.98$, $p<0.25$) and urine flow rate ($t=0$, $p<0.5$) were not statistically different between the renal dysfunction group (III) and the normal renal function group (I).

Hyperbilirubinemia was studied as a potential post-operative factor that could either correlate with or cause an increase in mortality from renal dysfunction. This condition appeared in renal dysfunction patients in a significantly higher incidence ($X^2=12.98$, $p<0.001$) than in the non-renal compromised patients, and correlates with total units of blood given during hospitalization (not with blood products administration during surgery) and with hypotension but does not correlate with any of the other factors described in the results.

Hyperbilirubinemia in shock patients has been evaluated in two studies in which abdominal aortic aneurysms were involved. One study[16] had 3 of 4 cases that were ruptured aneurysms and demonstrated on histology congestion, centrilobular degeneration, and mild fatty changes (a picture of obstructive jaundice). Total bilirubin levels here averaged 33.8 mgms/100ml and, in a paper on 8 cases of ruptured abdominal aneurysms,[15] total bilirubin averaged 28.4 mgms%--both much higher than the hyperbilirubinemia seen in this series. This would indicate that the jaundice seen in the current study was due to hepatic injury that was milder than the injury seen in the literature.

Mortality data in this series (Chart VII) emphasizes a great variance in pre- and intra-operative factors that can lead to ARF. In all ARF patients who died, a period of hypotension preceded the deterioration in renal function. All these patients had evidence of infection after surgery and all 3 patients who required dialysis died.

A "post-operative chronic renal failure" picture as described by Merion *et al*[20] was seen in only a single patient.

Conclusions

While the incidence of acute renal failure following abdominal aortic aneurysm resection was low in this series, the mortality rate, in accordance with other studies in the literature, was high. No single pre- or intra-operative factor in this study was found to be either predictive for or preventative of acute renal failure and, therefore, it must be supposed that the post-operative development of acute renal failure is a process that requires multiple clinical factors. The clinical setting that predisposes to this complication and the therapeutic measures that might prevent it need further definition.

CHART I

RENAL FUNCTION EVALUATED BY SERUM CREATININE AND BUN

	Group I	Group II	Group III	Group IV
Pre-Op Cr.	1.2±0.25(n=83)	2.2±0.58(n=7)	1.01±0.19(n=7)	1.7±0.40(n=7)
High Post-Op Cr.	1.2±0.29(n=89)	2.2±0.48(n=7)	1.6±0.20(n=11)	7.2±5.40(n=13)
Low Post-Op Cr.	0.9±0.24(n=71)	1.3±0.75(n=7)	1.0±0.22(n=10)	2.0±1.0(n=12)
Pre-Op BUN	18.7±5.8(n=89)	27.7±8.7(n=7)	18.1±9.7(n=11)	27.6±12.4(n=13)
High Post-Op BUN	21.5±6.9(n=88)	35.4±14.4(n=7)	31.7±9.0(n=11)	91.2±45.7(n=12)
Low Post-Op BUN	12.1±4.6(n=87)	19.1±10.3(n=7)	16.2±7.1(n=11)	26.8±14.0(n=12)

CHART II

INCIDENCE OF PRE-OP RISK FACTORS

	Group I (n=90)	Group II (n=7)	Group III (n=11)	Group IV (n=13)
Hypertension	29%(26)	71%(5)	64%(7)	46%(6)
Myocardial Infarction	36%(32)	29%(2)	9%(1)	38%(5)
Renal Disease	14%(13)	71%(5)	0%(0)	23%(3)
Liver Disease	4%(4)	0%(0)	9%(1)	0%(0)
Diabetes Mellitus	2%(2)	0%(0)	9%(1)	0%(0)
Emergency Resection	11%(10)	0%(0)	45%(5)	61%(8)
Deaths	1%(1)	0%(0)	0%(0)	46%(6)
Age (years)	67.6±7.9	71.8±8.1	66.3±6.2	70.4±7.8

CHART III

INCIDENCE OF INTRA-OP PARAMETERS

	Group I (n=90 if not noted)	Group II (n=7 if not noted)	Group III (n=11 if not noted)	Group IV (n=13 if not noted)
Anesthesia Time (minutes)	282±85(n=88)	288±79	287±125	354±128(n=13)
Aortic Clamp Time (minutes)	79±34(n=76)	90±73(n=4)	86±57(n=10)	96±47(n=10)
Mannitol Use	69%	57%	45%	61%
Neosynephrine Use	22%	43%	27%	61%
Units of Blood Products	6±4	5.4±3	7.2±3.5	13.3±10.0(n=12)
Total Urine Output (cc)	790±567(n=75)	540±344	725±623(n=10)	423±275(n=12)
Urine Flow Rate (cc/min)*	2.34 ±1.68(N=74)	1.7±1.2(n=7)	2.3±1.6(n=10)	1.27±0.84(n=12)
Renal Artery Compromise	3.3%	14%	18%	31%

*Urine Flow Rate calculated by Total Urine Output/Anesthesia Time for each n in the groups. These values were then averaged

CHART IV

HYPOTENSIVE PATIENTS

Pre-Operative Factors

	Groups I & II (n=32 unless noted)	Groups III & IV (n=17 unless noted)
Pre-Op Creatinine (mg%)	1.2±0.3 (n=29)	1.56±0.39(n=10)
Post-Op Cr--High (mg%)	1.3±0.4 (n=31)	5.59±5.4(n=17)
Wt/Ht Variance* (kg)	-2.98±9.42 (n=23)	-3.4±8.46(n=13)
Emergency Resection	4/32 = 12.5%	11/17 = 65%
Renal Disease	5/32 = 15.6%	2/17 = 12%
Myocardial Infarction	12/32 = 37.5%	2/17 = 12%
Hypertension	6/32 = 18.8%	9/17 = 53%
Mortality	1/32 = 3%	6/17 = 38%

*Values calculated from average difference (+) or (-) of Weight according to Height and Age Metropolitan Life Insurance Tables

CHART V

HYPOTENSIVE PATIENTS: OPERATIVE PARAMETERS

	Groups I&II (n=32 if not noted)	Groups III&IV (n=17 if not noted)
Anesthesia Time(min.)	276±99(n=31)	340±135(n=17)
Aortic Clamp Time (min.)	81±30(n=22)	98±52(n=14)
Mannitol Use	59%(19)	53%(9)
Units of Blood Products	5.9±3.7(n=32)	12.2±8.1(n=16)
Renal Compromise	0%(0)	24%(4)
Total Urine Output	784±465(n=28)	439±366(n=16)
Urine Flow Rate(cc/Min.)	2.4±1.4(n=28)	1.3±0.83(n=16)

CHART VI

HYPERBILIRUBINEMIA IN THE POST-OP COURSE

Incidence	15/97 = 15.5%	13/24 = 54%
Bili(D) - High (mg%)	1.87 ± 1.31 (n=14)*	1.96 ± 2.0
Bili(I) - High (mg%)	3.89 ± 2.1 (n=14)*	4.34 ± 3.7
Post-op Creatinine High (mg%)	1.43 ± 0.61 (n=15)	6.0 ± 5.9
Anesthesia Time (minutes)	328 ± 127 (n=15)	344 ± 146
Blood Products Given in Operating Room (units)	8.8 ± 5.5 (n=15)	11.3 ± 9.8
Blood Products (Total--units)	14.2 ± 8.6 (n=14)*	26.7 ± 14.9 (n=12)
Hypotensive	7/15 = 47%	10/13 = 77%
Previous Liver Disease	4/15 = 27%	0/13 = 0%
Emergency Resection	4/15 = 27%	8/13 = 61%
Mortality	0	3/13 = 23%

* One patient in this group had ascending cholangitis in the pre-operative period and had bilirubin D/I values of 27.1/13.3. This was the only patient in this group whose values were greater than 3.8/6.7 and, consequently, this case was not considered in the calculations.

CHART VII

PROFILE OF MORTALITY IN ARF PATIENTS

	Pt #1	Pt #2	Pt #3	Pt #4	Pt #5	Pt #6
PRE-OP FACTORS						
Age (years)	75	66	75	77	62	83
Rupture	+	+	+	+	-	-
HBP	-	-	-	+	+	+
MI	-	-	-	+	+	-
Renal Disease	-	-	-	-	+	-
OP. FACTORS						
Anesthesia Time (min)	400	235	345	560	570	300
Urine Total (cc)	320	400	110	1050	440	NR
Urine Flow Rate (cc/min)	0.8	1.7	0.31	1.9	0.77	NR
Blood Prod. (units)	10	NR	20	35	12	5
Mannitol	+	+	+	-	+	-
Neosynephrine	+	-	+	-	+	+
Renal Compromise	-	-	+	-	-	-
POST-OP FACTORS						
Total Blood Prod. (units)	24	45	NR	49	20	11
Sepsis	-	+	+	-	+	-
Penumonia	+	+	+	-	+	+
Tracheostomy	+	+	-	-	+	-
Jaundice	-	+	-	+	+	-
Oliguria	+	+	+	+	-	-
Dialysis	+	+	+	-	-	-
Cardiac Arrhythmias	+	+	-	-	+	+
Myocardial Infarction	-	-	-	-	+	-
POD of Demise	#14	#7	#36	#4	#14	#20
Cause of Death	Cardiac Arrest	Pneumonia Peritonitis*	Infected Graft*	Patent Suture Lines on Anastomosis*	Sepsis	Myocardial Infarction*

*Cause of demise determined by autopsy.

Bibliography

1. Abbott WM, Abel RM, Beck CH, and Fisher JE: Renal Failure After Ruptured Aneurysm. Arch. Surg. 110: 1110-1112 (Sept. 1975).

2. Abbott WM, and Austen WG: The Reversal of Renal Cortical Ischemia During Aortic Occlusion by Mannitol. J. of Surg. Res. 16: 482-489 (May 1974).

3. Abbott WM, Cooper JD, and Austen WG: The Effect of Aortic Clamping and Declamping on Renal Blood Flow Distribution. J. of Surg. Res. 14: 385-392 (May 1973).

4. Abel RM, Beck CH, Abbott WM, Ryan JA, Barnett GO, and Fisher JE: Improved Survival From Acute Renal Failure After Treatment with Intravenous Essential L-Amino Acids and Glucose. NEJM. 288: 695-699 (April 5, 1973).

5. Abel RM, Wick J, Beck CH, Buckley MJ, and Austen WG: Renal Dysfunction Following Open-Heart Operations. Arch Surg. 108: 175-177 (Feb. 1974).

6. Baue AE: Multiple, Progressive, or Sequential Systems Failure: A Syndrome of the 1970's. Arch Surg. (editorial) 110: 779-781 (July 1975).

7. Bhat JG, Gluck MC, Lowenstein J, and Baldwin DS: Renal Failure After Open Heart Surgery. Ann. Int. Med. 84: 677-682 (June 1976).

8. Casali R, Simmons RG, Najarian JS, von Haritizsch B, Buselmeir TJ, and Kjellstrand CM: Acute Renal Insufficiency Complicating Major Cardiovascular Surgery. Ann. Surg. 181: 370-375 (March 1975).

9. Chawla SK, Najafi H, Ing TS, Dye WS, Javid H, Hunter JA, Goldin MD, and Serry C: Acute Renal Failure Complicating Ruptured Abdominal Aortic Aneurysms. Arch. Surg. 110: 521-526 (May 1975).

10. Couch NP, Lane FC, and Crane C: Management and Mortality in Resection of Abdominal Aortic Aneurysms. Am. J. of Surg. 119: 408-416 (April 1970).

11. Darling RC: Ruptured Arteriosclerotic Abdominal Aortic Aneurysms: A Pathologic and Clinical Study. Am. J. of Surg. 119: 397--401 (April 1970).

12. Etheredge EE, Levitin H, Nakamura K, and Glenn WWL: Effect of Mannitol on Renal Function During Open-Heart Surgery. Ann. Surg. 161: 53-62 (Jan. 1965).

13. Evans C, Evans M, and Pollock AV: The Incidence and Causes of Post-Operative Jaundice. Br. J. of Anaesth. 46: 520-525 (1974).

14. Graham AL, Najafi H, Dye WS, Javid H, Hunter JA, and Julian OC: Ruptured Abdominal Aortic Aneurysm. Arch. Surg. 97: 1024-1031 (Dec. 1968).

15. Hermreck As, Proberts KS, and Thomas JH: Severe Jaundice After Rupture of Abdominal Aortic Aneurysm. Am. J. Surg. 134: 745-748 (Dec. 1977).

16. Kantrowitz PA, Jones WA, Greenberger NJ, and Isselbacher KJ: Severe Postoperative Hyperbilirubinemia Simulating Obstructive Jaundice. NEJM. 276: 591-598 (March 16, 1967).

17. Kennedy AC, Burton JA, Luke RG, Briggs JD, Lindsay RM, Allison MEM, Edward N, and Dargie HJ: Factors Affecting the Prognosis in Acute Renal Failure. Quart. J. of Med. (New Series XLII. 165: 73-86 (Jan. 1973).

18. Kleinknecht D, and Ganeval D: Preventative Hemodialysis in Acute Renal Failure--Its Effect on Mortality and Morbidity. Proc. of Acute Renal Failure Conference--1973. 165-185.

19. Kleinknecht D, Jungers P, Chanard H, Barbanel C, and Ganeval D: Uremic and Non-Uremic Complications in Acute Renal Failure: Evaluation of Early and Frequent Dialysis on Prognosis. Kid. Int. 1: 190-196 (1976).

20. Merino GE, Buselmeir TJ, and Kjellstrand CM: Postoperative Chronic Renal Failure: A New Syndrome? Ann. Surg. 182: 37-44 (July 1975).

21. Powers SR:, Renal Failure After Ruptured Aneurysm. Arch Surg. (editorial) 110: 1069 (Sept. 1975).

22. Stott RB, Cameron JS, Ogg CS, and Bewick M: Why the Persistently High Mortality in Acute Renal Failure? Lancet July 8, 1972. pp. 75-79.

23. Tilney NJ, Bailey Gl, and Morgan AP: Sequential System Failure After Rupture of Abdominal Aortic Aneurysms. Ann. Surg. 178: 117-122 (Aug. 1973).

24. Thompson JE, Vollman RW, Austin DJ, and Kartchner MM: Prevention of Hypotensive and Renal Complications of Aortic Surgery Using Balanced Salt Solution. Ann. Surg. 167: 767-778 (May 1965).

25. Van Heeckeren DW: Ruptured Abdominal Aortic Aneurysms. Am. J. of Surg. 119: 402-407 (April 1970).

26. Yashar JL, Indeglia RA, and Yashar J: Surgery for Abdominal Aortic Aneurysms--Factors Affecting Survival and Long-Term Results. Am. J. of Surg. 123: 398-405 (April 1972).

David Nanamaker 6 Hawthorne Terr. Branford Ct. 06405

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